

## Can Group B Streptococci Cause Symptomatic Vaginitis?

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### ABSTRACT

**Background:** Maternal cervicovaginal colonization with Lancefield group B streptococci (GBS) is an important risk factor for neonatal morbidity and mortality. About 15% of women are carriers of GBS. Usually, they are asymptomatic.

**Cases:** We describe two patients with symptomatic vaginitis for which no apparent cause was found. Both patients were heavily colonized with GBS. After antibiotic treatment, both became asymptomatic and culture negative, but after recolonization with GBS, symptoms resumed. This phenomenon was repeatedly observed. After emergence of resistance to antibiotics, local application of chlorhexidine appeared to be the only useful treatment.

**Conclusion:** We hypothesize that GBS-vaginitis may be a possible disease entity. Although at present it is not clear why some patients become symptomatic, we speculate that the immunologic response is somehow selectively hampered in such patients. *Infect. Dis. Obstet. Gynecol.* 7:206–209, 1999. © 1999 Wiley-Liss, Inc.

### KEY WORDS

vaginal discharge; vulvar maceration; chlorhexidine

Lancefield group B beta-hemolytic streptococci (group B streptococci [GBS] or *Streptococcus agalactiae*) play an important role in the development of neonatal morbidity and mortality. Infections that occur before the seventh day of life—so-called early onset infections—are usually caused by vertical transmission. Between five percent and 40% of women carry GBS. Carriers of cervicovaginal GBS are usually asymptomatic. However, one study showed that in patients with purulent vaginal discharge the prevalence of GBS was increased.<sup>1</sup> Colonization with GBS seems to be more prevalent in patients with excessive vaginal discharge.<sup>2</sup> A correlation has also been demonstrated between the diagnosis of vaginitis and colonization with GBS.<sup>3</sup> However, in the papers noted, there was no statement about GBS being the cause of the vaginitis or just a cofactor. We here describe two patients with

vaginitis presumably caused by GBS, a condition that, to our knowledge, has not been reported before.

### CASE REPORTS

#### Case I

A 39-year-old woman, mother of five children, reported abdominal pain, dyspareunia, and excessive vaginal discharge. She was first examined by a gynecologist, who diagnosed salpingitis and treated her with cefuroxime, metronidazole, and erythromycin. Although her symptoms disappeared initially, the vaginal discharge reappeared after a few days. She was then referred to our clinic. Excessive vaginal discharge and dyspareunia had been present for several months. Her medical history had been uneventful apart from endometriosis, for which she was treated with gestrinone, causing

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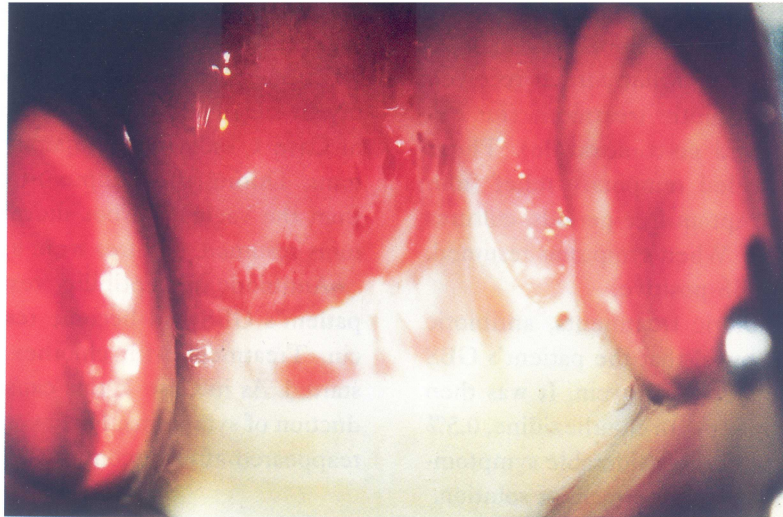


Fig. 1. Clinical picture of patient 1: abundant white/yellow discharge and erythema of vaginal walls.

amenorrhea. Childbirth had been uncomplicated each time, and she had had no cesarean deliveries. She had been married for a long time and had no other sexual contacts. In the preceding year, her husband had been hospitalized three times with periods of fever for which no explanation could be found. A culture from his rectum was positive for GBS.

Gynecological examination showed redness and maceration of vaginal introitus and perianal area (probably because of excessive vaginal discharge). There was abundant, watery, white/yellow, odorless vaginal discharge and erythema of vaginal walls (Fig. 1).

Laboratory examination included direct smears and cultures for *Neisseria gonorrhoeae*, *Trichomonas vaginalis*, and *Candida* species, all of which were negative. A culture for *Chlamydia trachomatis* was also negative. All of these tests were performed more than once, at different times. The Gram stain of vaginal discharge showed many gram-positive cocci and leukocytes (4+) (Fig. 2). Neither lactobacilli, clue cells, nor basal or parabasal cells were seen. An aerobic culture of vaginal discharge showed abundant growth of GBS. An anaerobic culture showed no growth of bacteria. Urinalysis, hematology, biochemistry, and serologic tests for

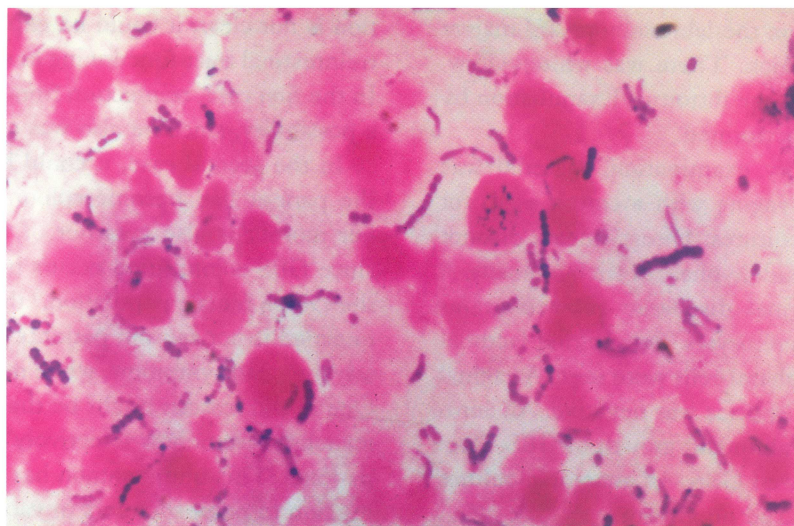


Fig. 2. Gram stain of vaginal fluid: streptococci and leukocytes.

syphilis and hepatitis B showed no abnormalities. A presumed diagnosis of GBS-vaginitis was made.

The patient together with her partner received several courses of antibiotic treatment (for example, clindamycin and co-trimoxazole). These treatments only provided temporary relief from symptoms. During symptom-free periods, cultures were negative for GBS. When symptoms resumed, cultures were again GBS-positive.

After several antibiotic treatments, antibiotic susceptibility tests showed that the patient's GBS had become resistant to clindamycin. It was then decided to start treatment with chlorhexidine, 0.5% gel. This antiseptic caused considerable symptomatic relief but did not offer a permanent solution.

### Case 2

A 19-year-old woman was referred to our clinic by a gynecologist because of persistent vaginal discharge, from which GBS was cultured at several occasions. Discharge had been present for about one year and was accompanied by vulvar and perianal irritation. She had already been treated with several antibiotics (e.g., penicillin) and antimycotics. All antibiotic treatments only caused temporary symptomatic relief.

A few weeks before she first visited our clinic, she had been hospitalized because of an episode of erythema nodosum for which no explanation was found. Otherwise, her medical history had been uneventful. The patient had never had sexual intercourse.

Gynecological examination showed maceration and linear erosions on the labia minora and majora and the perianal region. There was white/yellow, odorless discharge in the introitus. Examination by speculum was not performed.

For the laboratory examination, material was collected from the vagina with a cotton swab for direct smears and cultures. Direct smears did not show clue cells, pseudohyphae, or trichomonads. A Gram stain of vaginal discharge showed gram-positive cocci and leukocytes (2+); neither lactobacilli nor basal or parabasal cells were seen. A vaginal culture for *Candida* species was negative. An aerobic culture showed abundant growth of GBS. Rectal culture also showed GBS. An anaerobic culture of vaginal fluid showed no growth.

The patient's mother was also colonized with GBS. Routine testing for sexually transmitted dis-

eases was not done. A presumed diagnosis of GBS-vaginitis was made.

Local and oral antibiotic treatment (i.e., clindamycin cream and oral co-trimoxazole) resulted, as in Case 1, only in a temporary absence of symptoms. During the symptom-free period, cultures from the vagina did not show GBS. When symptoms resumed, cultures were again GBS-positive.

Antibiotic susceptibility tests showed that the patient's GBS had become resistant to clindamycin. Treatment with chlorhexidine gel was then started. As in Case 1, this caused considerable reduction of symptoms. However, symptoms quickly reappeared after discontinuation of treatment.

### DISCUSSION

In contrast with group A streptococcus, it is generally thought that GBS is not capable of causing vaginitis.<sup>4</sup> Our findings seem to suggest otherwise. Both patients described showed excessive white/yellow, odorless vaginal discharge, as well as vulvar and perianal maceration. This clinical picture, in combination with abundant presence of gram-positive cocci in the Gram stain and abundant growth of GBS in cultures, led to the hypothesis of GBS-vaginitis. This hypothesis is supported by the exclusion of other usual causes of vaginitis.

It is also supported by the occurrence of symptom-free and culture-negative periods after antibiotic treatment and the fact that reappearance of symptoms was associated with vaginal recolonization with GBS. Although the source of recolonization is not clear, it could be explained by the gastrointestinal tract functioning as a reservoir of GBS. In the first case, reinfection from the partner is also a possibility, since he also was a carrier of GBS.

In the second case, it could be speculated that the patient had become colonized at birth, since her mother is also a carrier of GBS. At any rate, since GBS colonization occurs in about 15–25% of the Dutch female population,<sup>6</sup> the most interesting question is not *how* our patients acquired GBS, but *why* they had symptoms, while most women do not. One of the reasons might be that the immunologic response is somehow selectively hampered in these patients. This phenomenon has been described in relation to nonresponders in vaccination programs. It is also frequently observed in patients with a selective deficiency of immunoglobulin G2 who

have recurrent infections with capsulated bacteria, such as meningococci.<sup>7</sup> It would be interesting to determine whether patients with symptoms of GBS-vaginitis have such deficiencies.

Treatment of GBS vaginitis appears to be a problem. Current literature only addresses attempts to decrease the rate of vaginal colonization in order to reduce vertical transmission.<sup>8</sup> Complete eradication of GBS-carriership is usually not a goal. Antibiotic treatment of GBS-vaginitis is probably hampered by the fact that recolonization is so easily established. The experience with our two patients seems to suggest that oral as well as local antibiotic treatment only has a temporary effect and might possibly lead to resistance against certain antibiotics. Local chlorhexidine application — a treatment that is also used to prevent neonatal transmission of GBS — seems to be the only useful alternative so far.

In conclusion, our report seems to provide circumstantial evidence that GBS can cause symptomatic vaginitis in some women, although causation can not be definitively ascribed. Given the chronicity of the condition and the absence of effective treatment (at least so far), further study of the subject seems appropriate.

## REFERENCES

1. Regan JA, Klebanoff MA, Nugent RP. The epidemiology of group B streptococcal colonization in pregnancy. *Obstet Gynecol* 1991;77:604–610.
2. Farrag OA, Gawad AA, Antar S. Group B-beta haemolytic streptococcal colonization in women using intrauterine contraceptive devices. *Contraception* 1985;31:595–602.
3. Jensen NE, Andersen BL. The prevalence of group B streptococci in human urogenital secretions. *Scand J Infect Dis* 1979;11:199–202.
4. Ginsburg CM. Group A streptococcal vaginitis in children. *Pediatr Infect Dis* 1982;1:36–37.
5. Dillon HC, Gray E, Pass MA, Gray BM. Anorectal and vaginal carriage of group B streptococci during pregnancy. *J Infect Dis* 1982;145:794–799.
6. van Oppen AC, Gerards LJ, Fleer A, Feldman RG, Bruinse HW. Diagnosis and chemoprophylaxis of perinatal infections caused by beta-hemolytic streptococci from Group B. *Ned Tijdschr Geneesk* 1993;137:583–587.
7. Umetsu DT, Ambrosino DM, Quinti I, Siber GR, Geha RS. Recurrent sinopulmonary infection and impaired antibody response to bacterial capsular polysaccharide antigen in children with selective IgG-subclass deficiency. *N Engl J Med* 1985;313:1247–1251.
8. Adriaanse AH, Kollée LAA, Muyltjens HL, Nijhuis JG, de Haan AFJ, Eskes TKAB. Randomized study of vaginal chlorhexidine disinfection during labor to prevent vertical transmission of group B streptococci. *Eur J Obstet Gynaecol Reprod Biol* 1995;61:135–141.